CALIFORNIA
AND
WESTERN MEDICINE

VOLUME XXXI AUGUST, 1929 No. 2

ECLAMPTOGENIC TOXEMIA*

ITS MANAGEMENT

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ECLAMPTOGENIC toxemia, like the poor, is always with us. In spite of an immense amount of research and speculation little progress has been made in arriving at the fundamental factors underlying its origin and development. However, great progress has been made by specialists in the management of the disease, so that the mortality and morbidity have been consistently and appreciably lowered in various parts of the world. However, Litzenberg has pointed out that the mortality rate advanced 5 per cent between 1915 and 1925 in the registered area of the United States. When we attempt to explain these facts several factors seem to be of importance.

INFLUENCE OF BETTER INSTITUTIONAL CARE

The prenatal management of the pregnant woman is today far better in many communities than formerly. Institutions like the Cook County Hospital now have regularly attended prenatal clinics where the great bulk of the obstetrical patients are seen and examined regularly and carefully.

Separate institutions, devoted entirely to obstetrical work and serving as a model in technique for the general hospitals, have developed in the larger population centers.

These institutions have influenced the situation in several ways:

1. By giving increasingly good obstetrical care to the patients.

2. By setting up competition standards which other institutions are forced to meet.

3. By serving as training ground for the development of young specialists, good students, and well-trained nursing personnel.

4. By dignifying the science of obstetrics in the eyes of both the profession and laity so that its problems and dangers are considered seriously.

5. By having the general hospitals recognize the importance of proper accommodations for the obstetrician and his patients, and by giving a service comparable to that which is put at the disposal of the general and other surgeons.

In addition to the above factors, more time and effort are being expended in some of the medical schools on the teaching of obstetrics, and more and more of the schools are furnishing their students part time paid instructors for this purpose.

Hospitalization of obstetric patients has become almost the rule in the larger centers, and this obviously permits closer observation during labor and the early puerperium; and permits of more rapid and efficient surgical intervention when this becomes necessary.

DETERRENT FACTORS

In addition to these advantages that the modern woman possesses and which her sister of several decades ago did not possess, there are, however, certain disadvantages which she has to assume that were formerly less prevalent. The average woman of today, I believe, is less rugged physically and probably immunologically, than the woman of thirty or forty years ago. Their lives are more sedentary, and they are less exposed to those ordinary slight wound infections which tend to stimulate general immunity.

There are more surgeons who are technically capable of doing a cesarean section but who have no conception of the fundamental obstetrical principles underlying the indications for operation in a given case. These men are called in consultation on obstetrical cases frequently. They know how to do a cesarean section, but have no ability or experience with other forms of obstetrical operations. Naturally they take the easiest way out. The patients, in most instances, would be far better off under strict medical management. Too often the patient is operated in a general operating room of a general hospital with a personnel actively engaged in the care of acute osteomyelitis, mastoiditis, empyema, and other pus infections. There has developed also an unwarranted "furor operativa" in some parts of the country sponsored by well-trained obstetricians. Such men are able to carry out these operations with a minimum of complications, but such practice leads inevitably to disaster when attempted by younger and less skilled individuals. All of these factors help to govern the mortality and morbidity rate in eclamptogenic toxemia.

ETIOLOGY

With these facts in mind let us consider what we know about the etiology of eclampsia. We can practically disregard the various theories that have been advanced and which attempt to explain

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* Read before the California Medical Association in General Meeting at the Fifty-Eighth Annual Session, May 6-9, 1929.

its fundamental mechanism, all of which have been more or less discredited.

Several facts are accepted by all:

1. Eclampsia occurs only in pregnant or recently pregnant females.
2. It is associated with degenerative changes in the parenchymatous organs, chiefly the liver, kidney, heart, and brain; but the lesions are not constant.
3. Hard physical work and a diet rich in protein predispose materially to the appearance and aggravation of the physical signs and symptoms.
4. The disease is rarely seen before the sixth month of pregnancy.
5. As a rule, there is a marked increase in the coagulability of the blood.
6. In most of the nonlethal cases clinical recovery is remarkably rapid and complete.

From the above facts I have endeavored to develop a conception of what is the matter with these women, and to outline a treatment based on this that would seem to fulfill the requirements. There is nothing fundamentally new in this, but the factors involved I feel have not been sufficiently stressed in this connection.

It has been shown by Vaughn and others that the protein molecule is highly toxic when split by enzymes in a certain way and to a certain degree. Thus egg-white, casein, or any apparently harmless protein substance contains a powerful and dangerous radicle if split in a certain way. This toxin may, on the other hand, be made completely innocuous by further splitting into its end products. We believe that the eclamptogenic toxemia woman is intoxicated by the products of protein metabolism. Our conception of the pregnant woman can best be appreciated by examining the diagram which I have prepared.

We believe that the pregnant woman is intoxicated from three main sources:

1. From the fetus and placenta directly and by the metabolic changes brought about in the maternal organism by their presence.
2. From the endogenous protein metabolic waste products.
3. From the exogenous protein metabolic waste products.

As a defense against these split protein toxins, the pregnant woman has:

1. The kidney, which is by far the most important factor in their elimination.
2. The bowels, which by stimulation can be made to assume part of the burden of excretion.
3. Blood-letting, which may play an important rôle under certain conditions.

The level of toxins in the blood at any given time depends on the ratio between intake and output of toxins. It is assumed that this level is constantly higher in the average pregnant woman than in the same woman when not pregnant, which may account for the slight and transient toxic symptoms so frequently seen in normal pregnant women, at or near term.

In considering the etiology of the disease we must further consider the harmful effect on the mechanism of toxin elimination and neutralization that is probably produced by the serious lesions found in the kidneys and liver, and the general depressant effect which pathologic changes in the brain and in the cardiac system must have on the vital processes. It is apparent that once these deleterious effects are produced a vicious cycle is started. The more the liver and kidney, heart and brain are damaged the more toxin accumulates in the blood, and the more toxemia the greater the damage to these organs.

PLAN OF TREATMENT

Any management, therefore, suggested for the treatment of eclamptogenic toxemia must aim at:

1. The reduction to the minimum of the influx of protein metabolic split products to spare the kidney especially.
2. The elimination as rapidly as possible of the toxic elements circulating in the blood in order to reduce the damage to the brain, heart, and liver.
3. The protection of the patient from injury during unconscious periods.
4. The support of the cardiac and respiratory centers until the peak of the intoxication is passed.

In addition to these factors we must consider the best way to meet the obstetrical problem involved, namely, the delivery of the baby, so that both the mother and baby may have the best chances for life and health, and the mother for future pregnancies.

It is obvious, therefore, that the treatment of eclamptogenic toxemia is a complex, not a simple, problem, and depends for its proper solution on a careful estimation of the various factors, favorable and unfavorable, that present themselves in different patients, and not infrequently in the same patient under various circumstances. The fundamental principle to be stressed is, we believe, that the condition is a protein split product intoxication, and that the reduction or elimination of these products is the rational method of attack or approach in treatment.

TREATMENT GROUPS

To this end we have divided patients, as they present themselves, into six main groups; and while realizing that the grouping does not cover all possibilities, nevertheless feel that it is sufficiently comprehensive to serve our purpose:

1. Normal obstetrical conditions plus mild toxemia.
2. Normal obstetrical conditions plus rapidly advancing toxemia.
3. Normal obstetrical conditions plus fulminating toxemia.
4. Abnormal obstetrical conditions plus eclamptogenic toxemia.
5. Intrapartum eclampsia.
6. Postpartum eclampsia.

MORTALITY PERCENTAGES IN DIFFERENT METHODS

Theoretically, with the proper type of prenatal supervision, eclamptogenic toxemia should be a very mild disease perfectly under our control and rarely complicated by convulsions. If we have the

proper facilities for modern obstetrical practice, death should be practically unknown from this toxemia.

The nearest approach to this standard, as reported by other writers, is the work of Stroganoff, who reports a series of two hundred and thirty personally treated cases in which the mortality was 1.7 per cent, he using his well-known expectant line of treatment. Edler, Lundquist, and others, using the Stroganoff method, report 8.5 to 20.2 per cent maternal mortality. Greenhill reports the results of cases treated by De Lee and his associates at the Chicago Lying-in Hospital, many of whom were treated actively by methods such as forceps, version, and cesarean section with a maternal mortality of 7.7 per cent.

Lazard, McNeile, and Vruwink and their associates have reported very good results in the management, especially of the convulsive stage of the disease, by the use of *magnesium sulphate* intravenously, as recommended and used by H. Einar. They use 20 cubic centimeters of a 10 per cent solution intravenously every two hours for six doses, the intervals between injections being lengthened as the patient improves. Blood pressure readings hourly are used as an index for more injections. These injections are combined with small doses of morphin or chloral and bromid per rectum. If convulsions are not imminent, 2 to 4 cubic centimeters of a 50 per cent solution may be given intramuscularly into the buttocks, two to four times a day. They were able to reduce the mortality in the neglected and desperate cases entering the Los Angeles County Hospital from 35 to 16 per cent. The *modus operandi* of this remedy is, according to Vruwink, not clear. Studies by Stander of Johns Hopkins tend to show that the method is not without some danger, if the magnesium sulphate is given too rapidly or in too concentrated solution.

Liver extract injections have recently been used by Mitler and Martinez of Pittsburgh on the supposition that, because of liver tissue destruction, the liver's normal detoxifying function was diminished, and this could be augmented and replaced by injecting liver extract. This theory we feel to be invalid because, first, there is no proof that the toxins of eclampsia can be neutralized by liver extract, or, second, that a detoxicating substance, if present in the liver cells, can be extracted in active form. Our thought is that quite the reverse might be expected, and that the toxemia might be augmented by the injection of a protein substance into a system flooded with proteolytic ferments. These writers, however, report a mortality of 6.9 per cent in only forty-three cases.

Titus and Givens have felt that the injection of *sugar solution* intravenously has a beneficial effect by supplying the immediate metabolic needs of the patient and by replenishing the depleted glycogen stores in the liver and muscles. We have not noted beneficial results from this management when used by others, and we have not adopted it.

What we wish to emphasize in this connection is the importance of the few fundamental principles that we are all familiar with in connection with this disease. Also how unnecessary a complicated theory as to its origin or treatment is, in order to get good results in treatment. In a broad sense this is true of practically all diseases. Typhoid, rabies, diphtheria, tuberculosis, and many other diseases could be wiped out of existence if we applied thoroughly the principles we already know for their eradication. We are firmly convinced that eclampsia can and should be added to this category.

In recent years a very decided change has come about in the attitude of most obstetricians toward the *operative management* of serious eclamptogenic toxemias. This has, in great part, been due to the above mentioned results which were obtained by Stroganoff and his school. Series as low as 8.5 per cent mortality in cases collected from several clinics using Stroganoff technique have been reported. A radical attitude in the management of these cases has become heretical in the minds of many. This view is based on the almost universally improving statistics in recent years and the concomitant increase in conservative management. We feel that there is some justification for this view, but believe that there are some other factors, such as better prenatal care, more hospital facilities and, on the whole, better trained obstetrical men throughout the country. These combine to make a radical difference in the way these patients are managed before, during and after delivery. For example, at the Cook County Hospital the mortality in patients diagnosed as having eclamptogenic toxemia has for years been between 25 and 35 per cent. In the last six years, with practically the same management in every other way, a prenatal clinic was added. The mortality has dropped to 12 per cent, and it is my firm belief that, with absolute control of the patients, many of this last group also could be saved. Indeed in my clinic across the street at the Illinois Research Hospital, we have had no deaths in over two hundred and fifty cases.

TREATMENT DETAILS IN DIFFERENT GROUPS

How, then, shall we meet the specific and practical problems that present themselves in connection with this disease. The first point of emphasis is the rigidity of the discipline and importance of attention to detail. The treatment may perhaps be best considered by using the grouping previously outlined.

A. *On the management of patients coming under Group 1 (with normal obstetrical conditions and mild toxemia and gestation not longer than thirty weeks):*

1. Bed rest, in hospital if possible, on milk diet, limited to 1000 cubic centimeters if much edema.
2. Magnesium sulphate one ounce every six hours until watery bowel movement.
3. Urine analysis, twenty-four-hour specimen, and quantitative albumin, phenolsulphonephthalein test.
4. Blood count, red, white and hemoglobin, blood chemistry and systolic and diastolic blood pressure.
5. Eye-ground examination.
6. Daily body weight.

If the symptoms gradually improve we add:

7. Fruits, vegetables, cereals, butter and bread, but prohibit tea, coffee, alcohol, meat, eggs, and fish.

Improvement continuing:

8. Sitting up in room and walking around.

TABLE 1.—*Résumé of Cases of Eclamptogenic Toxemia.*

	Preëclamptic	Nephritic
Number of cases	207	46
Convulsions	13—5.2%	3—6%
I. Age. Oldest	45	44
Youngest	14	20
Average	25	31.9
Optimum	15-20	35—40
II. Parity one	108	6
Two	22	4
Three	20	5
Over three	31	28
III. Pos. Wassermann	10	1
IV. General Symptoms.		
Headache	102	32
Edema	118	31
Eye	42	24
Epigastric pain	59	21
Vomiting	72	24
Convulsions	13	3—6%
V. Blood Pressure.		
Highest	235	278
Lowest	104	128
Average	163	182
VI. Urinalysis.		
Albumin	118	47
Sugar	9	4
Red blood cells	44	13
Casts. Hyaline	60	27
Granular	65	30
VII. Phenolsulphonephthalein.		
Reported cases	71	35
Lowest	5%	8.1%
Highest	65%	65%
Average	32%	30%
Optimum	30-35%	25—30%
VIII. Eye grounds.		
Reported cases	16	11
Normal	10	6
Retinitis bilateral	4	3
Retinitis unilateral	1	1
Sclerosis		1
Enlarged veins	1	
IX. Blood loss.		
Greatest	2000cc.	700cc.
Least	10	50
Average	250	211
Optimum	150	100
X. Treatment.		
Venesection	14	7
Induction of labor	72	22
Milk diet	162	38
Bed rest	151	36
Mg SO_4	106	40
Morphine	73	14
Saline	4	2
XI. Induction of labor.		
Bag	41	18
Quinin, castor oil	40	3
Rupture of membranes	5	1
XII. Type of delivery.		
Spontaneous	158	33
Forceps	19	1
Cesarean	9	7
Breech	5	
Version	6	5
Craniotomy	2	
XIII. Results.		
Maternal deaths		
Fetal deaths	26-12%	22—47%
Deformities	3	
Macerated	6	3
Under 2000	12	10
Viable babies	5—2.4%	9—19.5%

If at or near term, quinin and castor oil induction is tried, failing a slight increase in diet and exercise under close supervision.

If symptoms get worse under this regimen the management becomes the same as under Group 2.

B. *On the management of patients in Group 2 (rapidly advancing toxemia with normal obstetrical conditions):*

1. Quinin and castor oil, Watson method; failing.

2. Voorhees bag induction.

3. Termination of second stage by forceps or version and extraction; if complications ensue or are threatening.

4. Encourage postpartum hemorrhage up to 500 cubic centimeters; no pituitrin or ergot in third stage.

5. Morphin grain one-fourth hypodermatically as soon as the baby is born.

6. Diet of milk until blood pressure falls to 140 to 150 systolic, and urine begins to clear.

7. Magnesium sulphate intramuscularly or intravenously, depending on the severity of the symptoms, speed of appearance and progress.

C. *On the management of patients in Group 3 (fulminating toxic symptoms, normal obstetrical conditions):*

(a) *Ideal obstetrical surroundings:*

1. If not in labor, cesarean section; local or ethylene anesthesia especially indicated if the patient is a primipara.

2. If in first stage, cesarean section unless dilatation can be easily completed, when forceps or version may be substituted.

3. If in second stage, forceps or version, adding episiotomy; if a para one, morphin grain one-fourth after delivery of baby, and one-sixth every six hours afterward if necessary.

4. Five hundred to seven hundred cubic centimeters of blood are withdrawn from the cubital vein. The vein is best opened for this purpose. If operation is to be performed, bleeding is postponed until after the operative blood loss is known.

5. Magnesium sulphate 20 cubic centimeters of a 10 per cent solution given slowly intravenously.

6. If a multipara and markedly premature baby twenty-eight to thirty-four weeks, vaginal cesarean section may well be done.

(b) *If conditions are not ideal, due to poor hospital surroundings, inadequate help, infected patient, or incompetent operator, the Stroganoff method is advised, comprising:*

1. Morphin sulphate grain one-fourth hypodermatically as soon as patient is seen.

2. One hour later 20 to 40 grains chloral hydrate per rectum.

3. Two hours later morphin sulphate one-fourth grain hypodermatically.

4. Four hours later 30 grains chloral hydrate per rectum.

5. Six hours later 15 to 30 grains of chloral hydrate per rectum.

6. Seven hours later 20 grains of chloral hydrate per rectum.

The chloral is injected slowly in four or five ounces of warm water. The patient must be in a dark quiet room, and a light chloroform or ether anesthesia is given to prevent convulsions. Intravenous injections of magnesium sulphate may be used also as above. As soon as labor has advanced to second stage with descent, a forceps extraction may be done if the head does not advance. Unquestionably for the general man with inadequate facilities the Stroganoff method will give the best results.

D. *On the management of patients in Group 4 (abnormal obstetrical conditions plus eclamptogenic toxemia):*

Some of the more important conditions are:

1. Contracted pelvis of moderate or extreme degree.

2. Heart lesions with decompensation or recently established compensation.

3. Pulmonary disease, severe tuberculosis, pneumothorax, pulmonary edema.

4. Uterine fibroids, especially of the large obstructive type, and if patient is an elderly primipara.

5. Acute infectious diseases such as erysipelas, measles, and scarlet fever.

Each case must be individualized, and in general we may say that the earliest termination of pregnancy compatible with fetal safety, and by the most conservative method is the procedure of choice. With contracted pelvis and disproportion between the size of the baby and the pelvis, we lean toward cesarean section to avoid a long-drawn-out labor, with convulsions present, before its termination. Heart and lung lesions are best treated by cesarean section under local anesthesia for the same reason. If uterine fibroids offer insuperable obstruction, Porro cesarean is probably the best procedure, and if the toxemia is mild and the fibroids do not interfere with decent induction of labor, may solve the problem best. If acute infectious disease is present, avoid delivery if the toxemia is not of the fulminating type; and if it is fulminating do a Porro cesarean section, regardless. The indication for cesarean section in this group may be a double or a triple one. The reason we lean toward the radical side is the uncertainty of the ordinary forces of labor under the abnormal conditions present.

E. *On the management of patients in Group 5 (intrapartum eclampsia):*

Conservative measure may be tried, or:

1. In primipara, in the first stage of labor, cesarean section should be done unless the first stage can be easily completed, when forceps or version may be substituted. In multipara, dilatation may usually be accomplished by a bag, or manually.

2. In the second stage of labor, forceps or version, adding episiotomy if the patient is a primipara. Morphin grain one-fourth is given after delivery of baby, and one-sixth grain six hours afterward if necessary.

3. Late delivery room set up to avoid contamination.

4. Watch carefully by rectal examination to avoid precipitate delivery.

5. Prepare for asphyxiated baby, tracheal catheter and oxygen.

6. Limit examinations and operations to avoid infections.

7. Careful repair of wounds.

F. *On the management of patients in Group 6 (postpartum eclampsia):*

(a) *All cases of preëclamptic toxemia found before or during labor:*

1. Treated prophylactically in puerperium by magnesium sulphate, morning and afternoon by mouth, or 20 cubic centimeters of a 10 per cent solution intravenously.

2. Milk diet and bed rest.

3. Blood pressure three times a day until it stays below 140.

(b) *If convulsions:*

4. Venesection, 700 to 800 cubic centimeters.

5. Morphin, grains one-fourth or one-sixth, three times a day, if necessary.

6. Chloral hydrate, 20 to 40 grains in 4 to 5 ounces of water, per rectum.

7. Phenolsulphonephthalein test.

8. Protection against injury if convulsions arise. Injury to child or patient due to mental aberration. The danger period is in the first week of the puerperium. Symptoms rarely appear or persist in eclampsia thereafter.

(c) *The management of the eclamptic convulsions:*

1. Constant attendance of a trained person if possible.

2. Mouth gag to protect the tongue.

3. Sufficient help to prevent self-injury during convulsion.

4. Reduce all stimuli to minimum. Dark room, few examinations, and manipulations. Morphin and chloral are useful.

5. Bleeding is best done by venesection under local anesthesia.

RESULTS

By the application of these principles we have handled two hundred and sixty-five cases of eclamptogenic toxemia with or without convulsions. These have practically all been clinical cases in the charity beds of the University of Iowa and University of Illinois teaching hospitals. While the writer supervised most of the cases personally and did nearly all of the major operative work, the ordinary deliveries and the conduct of the labors were in the hands of his intern and residents. The prenatal supervision was excellent in almost all cases, very few having eluded the vigilance of our out-patient staff. Of these cases we have classified two hundred and twenty-one as eclamptics and forty-three as nephritic toxemias, on the basis of the evidence at hand, six weeks after de-

livery when the case was finally summed up. A concentrated résumé of the findings is given in parts one to seven of the table.

CONCLUSIONS

1. The mortality in eclampsia in the United States registered area is on the increase, in spite of numerous demonstrations in special clinics that the disease can be practically eliminated by efficient management.

2. Considerable evidence points to the fact that protein metabolic end-products play an important part in the etiology.

3. Limiting the production, and promoting the excretion of these substances, will in most instances prevent the development of serious symptoms.

4. When serious symptoms arise, in spite of prophylactic treatment, suggesting the probable onset of the dangerous convulsive stage, the pregnancy should be terminated as conservatively as the exigencies will permit.

5. Cesarean section is advised only in the fulminating type of eclamptogenic toxemia or in the presence of a border-line or actual indication for other causes, such as contracted pelvis, placenta previa, cardiac or pulmonary disease. Cesarean section should never be undertaken in eclampsia without, first, an accurate estimation of the obstetrical problem involved; second, a competent operator familiar with obstetrical operations; and third, proper assistance and surgical surroundings.

6. The eclamptogenic baby is toxic, frequently premature and below par, and careful attention should be given to its delivery, resuscitation and immediate postdelivery management.

7. When serious symptoms arise and operative intervention is contraindicated, the Stroganoff method of treatment is advised, combined with the use of magnesium sulphate intravenously if convulsive seizures supervene.

8. No great general reduction in mortality is to be expected until medical students, practicing physicians, and even the laity, have a clear understanding of these salient underlying principles.

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A PRACTICAL CONSIDERATION OF CHOLECYSTOGRAPHY*

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PUBLISHED statistics on the accuracy of cholecystographic diagnosis vary rather widely. Aside from differences due to the personal equation, the variation may be attributed to: (1) present inability of the surgeon, anatomist, physiologist, and histopathologist to distinguish minutely between the normal and abnormal gall bladder; (2) lack of agreement as to what constitutes the normal or abnormal gall bladder; (3) use or nonuse of clinical data in the making of cholecystographic diagnoses; and (4) variations in groups on which the statistics are based and in the methods of analysis.

Disease of the biliary tract is the most common abdominal lesion for which surgeons are called on to operate, and it is, therefore, of prime importance to determine who shall condemn or defend the gall bladder, whether the clinician, surgeon, roentgenologist, or pathologist.[7] Although the clinician carries the major portion of the responsibility in determining whether the gall bladder should be subjected to surgical exploration, nevertheless he depends on the roentgenologist and surgeon for help and advice, and eventually he appeals to the anatomist, physiologist, and pathologist for fundamental knowledge. The chain of diagnosis of cholecystic disease is, therefore, no stronger than its weakest link.

Cholecystography has brought to the attention of the anatomist the fact that the gall bladder varies greatly in form and position according to the habitus of the individual. Cholecystography also has aided the physiologist in his efforts to ascertain more specifically the functional behavior of the gall bladder and its alteration by nonpathologic states. Mann [11] and other physiologists consider the gall bladder to be a rather temperamental organ, susceptible of influence by many remote conditions. The work of Mann and Higgins [12] indicates that a degree of stasis occurs during pregnancy. Boyden[1] concluded from his studies that gall bladders of women empty more quickly than those of men. Plummer,[13] interested in the subject primarily as a clinician, has observed that a certain type of patient, characterized by being easily fatigued, by achlorhydria and by a low basal metabolic rate, is likely to respond eccentrically to

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* Read before the General Medicine Section of the California Medical Association at the Fifty-Eighth Annual Session, May 6-9, 1929.